WHY THE WORKPLACE AND CARDIOVASCULAR DISEASE?

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Cardiovascular disease (CVD) is the major cause of morbidity and mortality in the industrialized world.* While there have been trends towards lowered rates of CVD mortality in North America and Western Europe, CVD still represents a significant public health problem—indeed, a pandemic. In the former Soviet Union and other eastern European countries, CVD morbidity and mortality have increased dramatically over the last 30 years.[7] Rising prevalence rates also have been observed in many developing countries. Thus, "it has been projected that CVD worldwide will climb from the second most common cause of death . . . in 1990, to first place, with more than 36% of all deaths in 2020."[5]

In the U.S. alone, CVD is the cause of 41% of all deaths.[2] An estimated 250,000–350,000 people annually die suddenly of heart disease in the U.S.,[3,6,10,18,20,21] and at least the same number lose their lives more slowly due to manifest CVD from which they have chronically suffered.

The dominant focus of research and intervention in the medical community has been on **individual traits**, especially genetic susceptibility and risky behaviors (e.g., smoking, over-eating, sedentary lifestyle) as playing a primary role in the etiology of CVD. The underpinnings of this explanation of the CVD epidemic lie in the development of powerful engineering models. Modern advances in the physical sciences lend themselves particularly well to the study of the cardiovascular system. Namely, it has appeared that CVD could be characterized as a disturbance in hydraulic (hemodynamic) and/or electrical (electrophysiologic) function.[5] Coronary atherosclerosis (i.e., vascular obstruction) has been designated as the "prime mover" of cardiovascular disorders, such that a series of atherogenic risk factors were sought, and many were identified (e.g., hypercholesterolemia, hypertension, diabetes, obesity). According to this view, the CVD epidemic can be curtailed by: (1) better management of atherogenic risk factors, (2) use of available medical treatments and more technological advances, and (3) additional research into the molecular biology of atherogenic and other cardiodegenerative processes.

It is indisputable that this approach represents an invaluable advance in our battle against CVD. Millions of people have been protected by quitting smoking,

* Includes arteriosclerotic heart disease, cerebrovascular disease, and peripheral vascular disease (ICD codes 400–404, 410–414, 430–436, 440–445).

eating a "heart-healthy" diet, and exercising. And countless patients with manifest coronary heart disease (CHD) have been saved by percutaneous transluminal coronary angioplasty and coronary artery by-pass surgery, not to mention the life-saving armamentarium of pharmacologic agents we now have at our disposal. Cardiac pacemakers and automatic implantable cardioverter defibrillators provide hope for a normal life to many patients suffering from life-threatening cardiac rhythm and conduction disturbances. Only 50 years ago, nearly all of these patients would have been doomed.

Despite the optimism engendered by these achievements, we believe that a closer look at the overall public health impact of this traditional medical approach to CVD is in order. While these methods of electrical and hemodynamic systems are highly sophisticated, the etiology and pathogenesis of CVD cannot be reduced to a series of disordered pumps and electrical circuits. Furthermore, the intimate connections between the social environment and the central nervous system (CNS), and the CNS and the cardiovascular system via the autonomic nervous system, compel one to look beyond the cardiovascular system in isolation to fully appreciate how CVD develops.

In point of fact, both our understanding of the etiology of CVD and our ability to manage the epidemic are still limited. For example, the Framingham Heart Study used epidemiologic techniques to identify important risk factors (smoking, diabetes, hypertension, and cholesterol). However, these traditional risk factors explained only part of the risk for CHD.[11] In practical terms, this means that these standard risk factors fail to predict many of the new CHD cases. Note that one of these factors—essential hypertension—is of practically unknown etiology. Moreover, these traditional risk factors represent relatively "proximate" causes of CVD; each of them, in turn, has a complex set of determinants, many of which are of psychosocial origin.

New developments expand and challenge the focus on these traditional, proximate risk factors. One of these is the emergence of research into **behavioral factors** that might influence the development of CVD. A notable example has been the formulation of the concept of CVD-prone behavior—the Type A behavior pattern (TABP).[9] While initial study results from the Western Collaborate Group Study indicated that TABP was a strong independent predictor of CHD mortality,[24] subsequent research has failed to substantiate these findings.[22] More recently, hostility—a component of Type A behavior—has emerged as a possible risk factor for CVD.[30]

The importance of the TABP is not so much its contribution to the explanation of CVD, but its laying the groundwork for **social psychology** to examine the impact of the social and psychological environment on CVD. TABP was a stepping-stone to the investigation of the role of the workplace in CVD.

This formulation regarding TABP is complemented by a body of epidemiologic literature which documents the strong role of social experiences, beginning in childhood and extending through working life and beyond, in shaping human behavior. For example, recent research has demonstrated that characteristics of people's jobs, such as high or low decision-making authority, are associated with the development of specific complex behaviors and personality attributes.[4,14–16,27]

Another development that has expanded traditional cardiovascular epidemiology has been the field of **social epidemiology**, which examines factors such as social networks, social support, and social class as potential causes or modifiers of disease processes.[8,17,23,28] For example, social epidemiologists have demonstrated that lower socioeconomic status is an important risk factor for CVD.[12,19] Nonetheless, even with

the inclusion of these social and behavioral factors, there is still a large amount of unexplained variance in CVD, as well as in essential hypertension.

We wish to argue that to better understand the CVD epidemic, social epidemiology needs to incorporate, in a much more prominent manner, a heretofore relatively neglected realm of social life—the workplace. We briefly present the case of essential hypertension (EH) as an illustration of our argument. EH is a major risk factor not only for CHD, but also for left ventricular hypertrophy, stroke, renal disease, and many other major pathologic processes. This disease afflicts 60 million Americans and 600 million people worldwide. The identified risk factors (i.e., obesity, salt intake, genetics, age, alcohol intake) explain only a small part of the risk.

A social epidemiologic approach suggests that EH is a disease of industrialized society.[25] There is a minimal hypertension disease burden among hunter-gatherers, nonmarket agricultural communities, and other nonindustrialized societies.[29] Within industrial society, hypertension is socially patterned by class, race, ethnicity, urbanicity, and gender. Current evidence implicates the unidentified causes of EH as most likely to include one or more ubiquitous exposures, suggesting the need to examine diet, lifestyle, work, and community. An adequate explanatory risk factor also should incorporate the social patterning of the disease.

Hypertension as an epidemic seems likely to be of relatively recent historical origins. Work organization has changed profoundly during the past 200 years. Craftwork, which predominated for many centuries, was largely replaced by the industrial revolution. Skilled workers, who had exercised substantial control over their work processes, were replaced by lower-skilled labor in new machine-based production technologies.[13] At the beginning of the 20th century, Taylorism further reshaped the workplace with its emphasis on narrow performance and efficiency using the technique of the assembly line, at the expense of employee collectivity and broader employee expertise and knowledge of the work process. Even lower-level, white-collar work, through office automation, has been shaped by the principles of the assembly line. Small businesses have been replaced by large centralized, multinational organizations. Most importantly, power to control the production process has been increasingly concentrated in the hands of management. The recent trend has been toward an acceleration of these changes in the workplace, characterized by a system of work organization known as "lean production." "These dynamics include organization restructuring, mergers, acquisitions, and downsizing, the frantic pace of work and life, the erosion of leisure time and/or the blending of work and home time. Most of these developments are driven by economic and technological changes aiming at short-term productivity and profit gain."[1]

The contemporary work environment is the locus in which adults now spend the majority of their waking hours, performing activities which are increasingly characterized, both by scientists and the workers, as demanding, constraining, and highly stressful. We know that blood pressure (BP) is elevated during working hours. We also know that performing demanding, constraining and otherwise mentally stressful activity provokes sharp rises in BP.

As will be demonstrated in this volume, an emerging body of evidence implicates specific features of work as important causes of hypertension as well as other manifestations of CVD. Chapters 2–5 explore this empirical evidence and the theoretical constructs concerning the relationship between the workplace and CVD. Psychosocial factors identified with the workplace are a particular focus. We develop the paradigm of "econeurocardiology" as a conceptual bridge which renders plausible the various theoretical constructs of work stress as they relate to CVD. The

econeurocardiology concept offers a framework in which the reader can understand how these stress mechanisms give rise to various cardiovascular target organ responses. Chapter 6 explores the methodological issues in the measurement of psychosocial factors at the workplace. Recommendations are made on how to improve the reliability, validity, and feasibility of these measures. Chapters 7–10 offer the clinician a set of tools for the evaluation and management of working people at risk for heart disease. A new, more advanced approach to "occupational cardiology" is presented. Chapters 11–13 provide a public health overview, addressing economics and the legislative, legal, and preventive interventions necessary to deal with this workplace-induced CVD epidemic.

Finally, Chapter 14 summarizes the evidence, makes the case for a causal relationship between the workplace and CVD, and discusses the implications of the trends toward deteriorating working conditions (e.g., lean production, downsizing, and longer work hours). These trends may result in greater exposure to psychosocial risk factors at the workplace, which may, in turn, increase the CVD epidemic. Since this CVD epidemic is engendered, at least in part, by the social organization of work and other noxious workplace exposures, primary prevention may be possible via interventions aimed at improving the work environment. Legislative changes and public health interventions can help create a climate in which healthy work becomes the priority.

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